ON THE USE OF THE TEMPORAL FASCIA TO COVER IN CRANIAL DEFECTS.*

BY CARL BECK, M.D.,

OF NEW YORK,

Professor of Surgery in the New York Post-Graduate Medical School and Hospital; Surgeon to St. Mark's Hospital.

AMONG the manifold methods in use for covering over defects of the cranium, the osteoplastic, employing a living bone-flap, as advised by Mueller and Koenig, is the one most commonly selected. Only in cases where the execution meets such technical difficulties as insufficiency of diploe or excessive thinness of the surrounding material, would one fall back upon one of the various heteroplastic procedures. In these subsequent remarks may it be permitted me to point to another possible method of bridging over the defect. This is not a technical improvement over those most excellent methods already mentioned, but at the same time it meets an indication in lacerated wounds which they do not, viz., the question of adhesions after cicatrization. And indeed this point is of decisive import. Jackson, as is well known, noticed that cerebral epilepsy can be caused by minor tissue changes, especially scars. The recognition of this etiological fact nurtured the thought of removing parts of the scar, or similar changes. Several operators have actually been successful in achieving results in this hitherto unfertile field.

The report of Graf[1] comprises 92 cases of Jacksonian epilepsy, in 82 of which tissue changes had taken place. These involved either the brain, the membranes of the brain, or the skull itself. Seventy-one cases in all were operated upon; 4 died, 20 remained unimproved, but 22 were completely cured. Furthermore it appears that 23 cases which had been observed for too short a time to justify a final verdict became well. For it is often the case that the attacks vanish for a space of time, due perhaps to transient traumatic irritations, and then recur later on with renewed or aggravated violence.

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* Read before the German Medical Society of New York, April 30, 1906, with presentation of patient.

[1] Operative Behandlung der Epilepsie, Arbeiten aus der chirurgischen Klinik der K. Universitaet, Berlin, 1898, Thl. 13.

The best surety for permanent cure seems to me to prevent firm reunion after extirpation of the cicatrix. The apposition of periosteum and brain must necessarily cause more or less unyielding scars. A plastic transposition of the dura can only be thought of in small defects. Therefore I sought a material which shows no marked tendency toward adhesion, and believe I have found it in the strong fibrous sheath presented by the temporal fascia. We know that in operations upon aponeuroses, union takes place with the surrounding structures through loose, wide-meshed, web-like connective tissue. When we fold over the flap gained from the temporal muscle and periosteum, then the brain is covered by fascia, the periosteum remaining on top. Slowly a protecting lid is formed which gives ample protection even though it does not attain the thickness of a bone-flap. In this wise the question of cover and adhesion is considered at one and the same time. In the following case the theoretical reflection has been borne out in practice.

The patient, 41 years old, had been well until he fell from a height of 20 feet on May 6, 1904. He landed on stony ground and in an unconscious state was carried off to a hospital where he lay in a comatose condition for eleven days.

As far as I can find out, the diagnosis of fracture of the skull had been made, and several bone-splinters had been removed on the day of the injury. Twelve days later the wound had healed and the patient was allowed to go home. He was treated there by his family physician Dr. W. A. Goodall, from whose report I gather that the right arm and leg showed slight signs of paralysis during the first three weeks after his discharge from the hospital. These gradually grew better until, six weeks after the injury, epileptic attacks set in. These came on without the usual aura. Patient suddenly became unconscious, the fingers, arms, and then the legs began to tremble. Gradually relaxation set in and patient slowly awoke from his senseless state. During these attacks, which lasted from three minutes to three hours, the pupils failed to react. There was also a marked psychical change in patient. Memory had grown weaker; formerly good-natured, he became morose and easily irritated. Patient's wife declares that his intelligence has suffered. There seems to have been no injury to the substance of the brain. Alcohol is no longer well borne, as the use of same causes congestion.

In September, 1905, I had the first opportunity of seeing the patient. At the time I found an elliptical defect, about three inches long and one inch wide at its greatest diameter, which reached from the summit of the left frontal bone to the temporal. Compare with skiagraph Fig. 4, which shows the shape of defect slightly enlarged. The gap was very large and showed distinct pulsation. No bone substance could be found in the groove.

It appears as though the skin had grown directly onto the cerebrum. A skiagraph, taken a short while later, confirmed the total absence of bone-substance.

In order to create a protection for the gap and also to prevent the formation of adhesions, I proposed an osteoplastic operation to the patient. He consented very much later after having undertaken numerous consultatory travels. Three months ago he entered the Post-Graduate Hospital where Prof. Hammond was kind enough to subject him to a neurological examination. He agreed with me in the belief that we had to deal with a case of Jacksonian epilepsy. Operation seemed especially indicated as the attacks had lately become not only more violent, but the disturbances in the function of the senses had also progressed.

On February 8th I laid bare the defect with an incision which passed around the gap in a wide circle. Slowly working my way along from the side I divided the scar tissue, holding the cutting edge of the knife toward the skin so as not to injure the brain. Then I found that the dura had remained intact

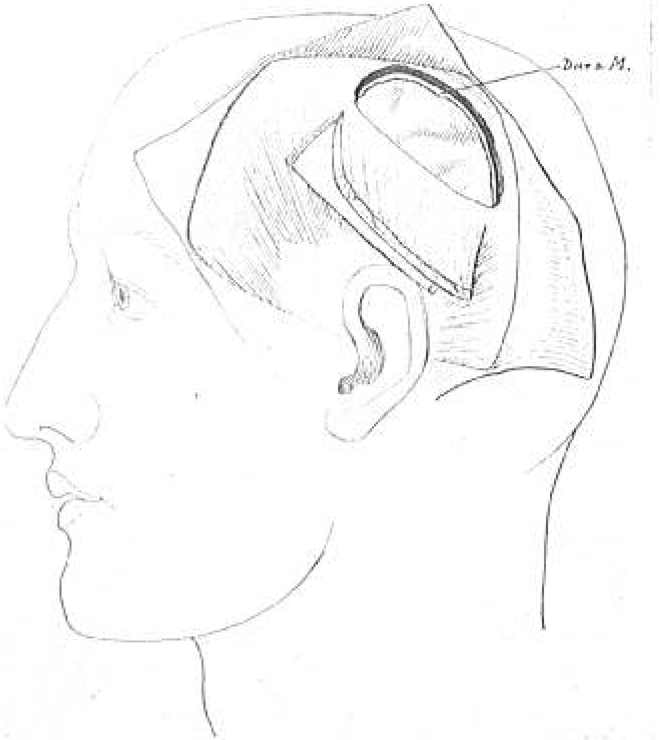

FIG. 1.—Flap of temporal fascia being raised adjacent to cranial defect.

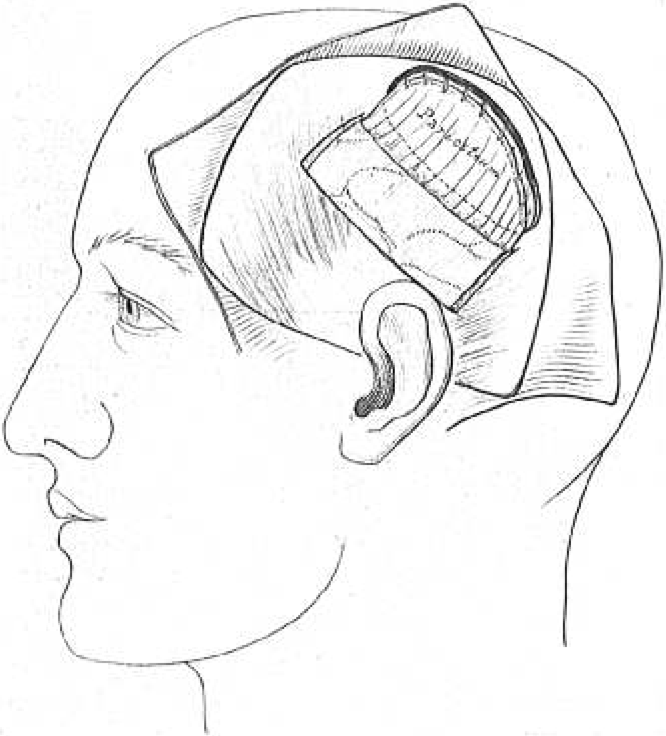

FIG. 2.—Flap of temporal fascia reflected so as to cover brain surface exposed by cranial defect.

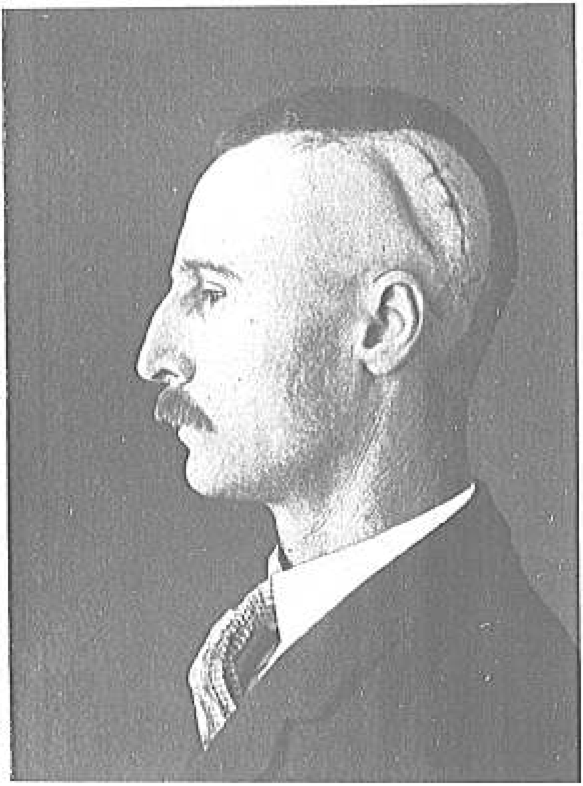

FIG. 3.—Showing appearance of patient three months after plastic operation to close cranial defect.

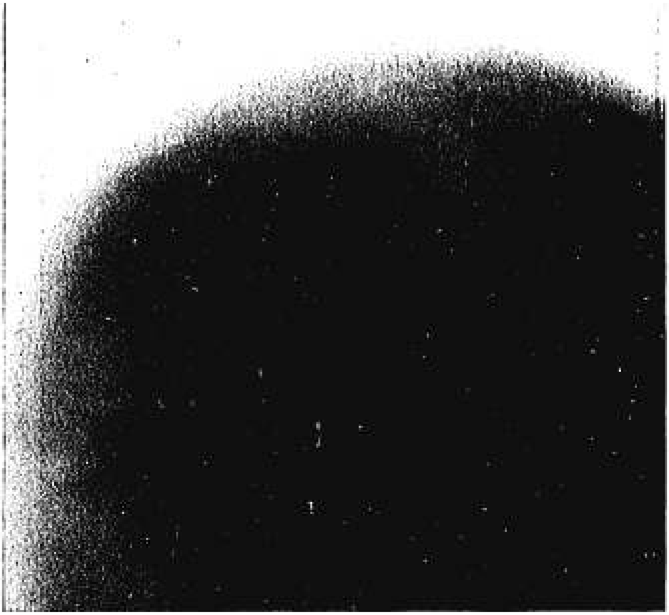

FIG. 4.—Showing bone proliferation in the lower two-thirds of the defect three months after plastic operation.

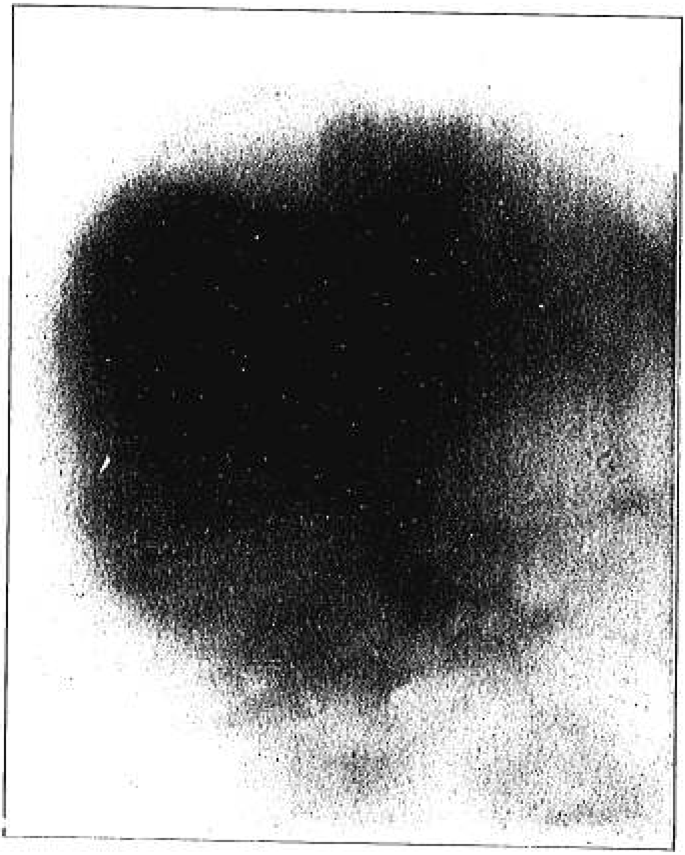

Fig. 5.

only along the edge of the defect. As the skiagraph had shown, small stalactite-like projections had formed at different spots along the edge. These were removed by scissors and bone-shears. During this manipulation a small cyst which had formed under one of these protrusions was emptied of its greenish-yellow contents.

Now, if, as apparent, the pressure-symptoms were principally the result of the extensive scar-formation, then it was necessary to employ a method in covering the defect, which excluded such dense adhesions as had formed. As already mentioned I believed the needed material would be found in the temporal fascia. I commenced the procedure by carrying an incision around the muscle down to the bone. Then I loosened the flap with the periosteum, taking great care not to tear the connecting link of periosteum which was kept as close to the bone suture as possible. (Fig. 1.) Although the periosteum was firmly adherent there it was but loosely attached to the rest of the bone and might easily have been torn off, a condition which might have ruined the whole procedure.

I folded over the entire flap at the lower edge of the defect and covered the gap with it. At the opposite end I chiselled off enough bone to enable me to make use of a small amount of dura to which I attached the aponeurotic end with thin catgut sutures. (Fig. 2.) The aponeurosis then lies in direct contact with the brain, with the periosteum on top. The skin surfaces were united. The wound healed primarily. The general health of the patient has improved and he feels normal in every way. Up to date he has had no convulsion.

I am well aware of the fact that the period of observation has been too short to make a pass verdict concerning the convulsions, and I will report concerning them again later on.

As far as the plastic operation is concerned, a decided success has been gained. Although the operation enlarged the defect somewhat by the removal of bone, the defect at present shows a smaller gap than before (Fig. 3). The latter is not as large as before operation. If we palpate the groove we can plainly demonstrate the bone which has formed from the periosteum. The same is visible on the skiagraph (Fig. 4) as a faint cloud. Of course the lid is not as solid as a bone-flap would make it. But is this absolutely a necessity, and does not this light covering perhaps render the same service? The utilization of aponeuroses in the place of muscle-tissue might also prove of value in such cases in which we desire to prevent reunion by insertion between bone fragments (as anchylosis, etc.).

Since making this report I have had another opportunity of performing the same operation on a boy of thirteen years, who fell out of a window a year ago and sustained a compound fracture of the skull. I am informed that he vomited considerably during the first twenty-four hours, was paralyzed, but able to talk. Ever since he showed symptoms of Jacksonian epilepsy. May 13, I exposed the defect the size of which exceeded somewhat that of a silver dollar, at the St. Mark's Hospital. There were dense adhesions between skin, dura and arachnoid, which were divided. The defect was situated on the left parietal bone. As it extended nearly up to the top of the skull, it required a long flap to reach it. There was no reaction after the operation, the patient being well at the present writing (two weeks after the operation).